SUPREME COURT OF THE STATE OF NEW YORK
COUNTY OF NEW YORK

IN RE: NEW YORK CITY ASBESTOS LITIGATION	NYCAL
The Document Relates To:	I.A.S. Part 11 (Madden, J.)
FRANK BIANCO, et al., Plaintiff(s), -against- A.O. SMITH WATER PRODUCTS, et al., Defendants.	Index No. 115546-06

SUPPLEMENTAL AFFIRMATION IN SUPPORT OF PLAINTIFFS' MOTION, PURSUANT TO FRYE V. UNITED STATES, AND *IN LIMINE*, 1) TO PRECLUDE THE NOVEL, UNSCIENTIFIC, NOT GENERALLY-ACCEPTED, LITIGATION-BASED, PREVIOUSLY EXCLUDED ASBESTOS "DOSE RECONSTRUCTION"/"EXPOSURE ASSESSMENT" ANALYSES AND ALL TESTIMONY RELATED THERETO, AND 2) TO PRECLUDE THE CASE-SPECIFIC REPORTS AND RELATED TESTIMONY OF DRS. RABINOVITZ AND WEINBERG SPECIFICALLY AS IMPROPER

STATE OF NEW YORK)
COUNTY OF NEW YORK) S.S.:
CITY OF NEW YORK)

THOMAS COMERFORD, ESQ., being duly sworn, deposes and says:

1. I am an attorney at law duly licensed to practice in the State of New York and before this Court, and I am a member of the law firm of Weitz & Luxenberg, P.C., attorneys for the above-captioned plaintiffs. As such, I have personal knowledge of the facts stated in this affirmation.

2. This supplemental affirmation is submitted in further support of Plaintiffs' motion, pursuant to Frye v. United States, and *in limine*, 1) to preclude the novel,

unscientific, not generally-accepted, litigation-based, previously excluded asbestos "dose reconstruction"/"exposure assessment" analyses and all testimony related thereto, and 2) to preclude the case-specific reports and related testimony of Drs. Rabinovitz and Weinberg specifically as improper, dated August 21, 2007.

3. These nine mesothelioma actions are for personal injury/wrongful death sustained by Plaintiffs as a result of their exposure to asbestos from the defendants asbestos-containing products and are scheduled for trial before Your Honor on September 12, 2007.

4. Plaintiffs herein submit Kentile Floors, Inc.'s (hereinafter "Kentile" or the "Defendant") Designation of Expert and Trial Witnesses list (attached hereto as **Exhibit A**) and Kentile's expert, Dr. Howard M. Sandler, M.D., reports regarding plaintiffs Harvey Helfand (attached hereto as **Exhibit B**) and Jack Nacht (attached hereto as **Exhibit C**), all served on Plaintiffs counsel on August 24, 2007.

5. Plaintiffs incorporate herein previous arguments briefed in their Affirmation and Memorandum of Law, dated August 21, 2007, and further object to any "dose reconstruction"/"exposure assessment" language found within Kentile's expert report submissions.

6. Specifically, such dose reconstruction/exposure assessment language is found in the C.P.L.R. § 3101(d) blurb regarding Kentile proposed expert witness Charles Redinger.[1]

[1] *Kenrile Floors, Inc.'s Amended Designation of Expert and Trial Witnesses*, August 22, 2007 (attached hereto as **Exhibit A**)("He will discuss...how the different characteristics of these different types and sizes of fibers affected the dose of respirable asbestos to which each plaintiff may have been exposed)(emphasis added).

7. Additional dose reconstruction/exposure assessment language is found in Kentile expert, Dr. Howard M. Sadler, M.D., expert reports regarding Plaintiff Harvey Helfand[2] and Jack Nacht.[3]

8. WHEREFORE, the Plaintiffs respectfully request this Honorable Court issue an order precluding any of the Defendants from admitting any documentary evidence or testimony regarding dose reconstruction/exposure assessment estimates at trial, and further, specifically excluding Kentile's proposed expert witnesses, including Dr. Charles Redinger and Dr. Howard Sadler, from testifying to same.

Certification Pursuant To 22 N.Y.C.R.R. § 130-1

9. I hereby certify, pursuant to 22 N.Y.C.R.R. § 130-1.1-a(b), that to the best of my knowledge, information and belief, formed after an inquiry reasonable under the circumstances, the presentation of papers listed below or the contentions therein are not frivolous as defined in 22 N.Y.C.R.R. § 130-1.1-(c).

Dated: New York, New York
September 7, 2007

Thomas M. Comerford

[2] *Howard M. Sadler, M.D., Expert Report RE: Harvey Helfand*, August 21, 2007 at 13 (attached hereto as **Exhibit B**)(discussion and application of dose exposure assessment studies, including utilization of the terms "time weighted average exposures" and "f/cc," to Plaintiff Helfand's exposure narrative).

[3] *Howard M. Sadler, M.D., Expert Report RE: Jack Nacht*, August 22, 2007 at 11-12, 20 (attached hereto as **Exhibit C**)(discussion and application of dose exposure assessment studies, including utilization of the terms "time weighted average exposure level," "f/cc," "Threshold Limit Value," "lifetime asbestos exposure," and "fiber year cumulative dose" to Plaintiff Nacht's exposure narrative).

EXHIBIT A

SUPREME COURT OF THE STATE OF NEW YORK
ALL COUNTIES WITHIN NEW YORK CITY

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IN RE: NEW YORK CITY ASBESTOS LITIGATION

This Document applies to:

ALL MAY 2007 CASES BEFORE JUSTICE JOAN MADDEN WHERE KENTILE FLOORS, INC. IS A REMAINING DEFENDANT

Jack Nacht Index No. 114274/06
Harvey Helfand Index No. 117176/06
James Director Index No. 115923/06

KENTILE FLOORS, INC.'S AMENDED DESIGNATION OF EXPERT AND TRIAL WITNESSES

KENTILE EXPERT AND TRIAL WITNESSES

Defendant KENTILE FLOORS, INC. (hereinafter "KENTILE"), by and through its attorneys, HARRIS BEACH PLLC hereby designates the following expert and trial witnesses, and hereby joins in all other Defendants' Designations of Expert and Trial Witnesses.

KENTILE reserves the right to use and call at the time of trial any expert, physician or fact witness designated by plaintiff.

KENTILE reserves the right to call any and/or all of the following persons as witnesses at the trial of this case, either live or by deposition, including:

1. Any custodian of records for any and all named defendants and third-party defendants to authenticate any and all records or documents which may be used in these cases;

2. Any expert or physician listed by plaintiff, any defendant, or dismissed defendant, who is expected to testify about asbestos-related disease, including any such expert who has reviewed x-rays or issued a report on the medical condition of the plaintiff in this case;

3. Any expert witness designated by plaintiff, defendant or any dismissed defendant, including the statement of subject matter, opinion and grounds of that witness;

4. Any treating, diagnosing physician, or other healthcare provider, of the plaintiff;

5. Any witnesses listed or called by plaintiff, or any other party, including any dismissed parties;

6. Any Representatives of any of plaintiff's employers or companies on whose premises plaintiff worked;

7. Any and all personnel referenced in plaintiff's employment records;

8. Any co-workers, employers, supervisors, foremen or superiors of plaintiff;

9. Representatives and/or records custodians of plaintiff's employers and of any companies on whose premises plaintiff worked;

10. Any records custodian necessary to authenticate and offer in evidence any records obtained by subpoena;

11. Any person identified in any party or former parties answers to discovery or deposition;

12. Any and all parties or dismissed parties to this litigation;

13. Any representatives and/or records custodians of all co-defendants;

14. Any representatives and/or records custodians for each and every physician, hospital, and health care provider for plaintiff;

15. Any representatives and/or records custodians of the Internal Revenue Service regarding records with information about plaintiff;

16. Any representatives and/or records custodians of the Social Security Administration regarding records with information about plaintiff;

17. Any representatives and/or records custodians of any accident, health, life and/or disability carrier for plaintiff regarding records with information about plaintiff;

18. Representatives and/or records custodians of the Worker's Compensation Fund regarding any records with information about plaintiff;

19. Any family members, friends, or relatives of plaintiff;

20. Any and all physicians, nurses and other medical personnel referenced in any of plaintiff's medical records;

21. Any person needed for the authentication of any documents, medical records or other evidence;

22. Any fact and/or expert witness necessary for rebuttal;

23. Any other witness necessary to publish demonstrative evidence to the jury;

24. KENTILE reserves the right to use any exhibit introduced by any party to this action or to comment upon any items prepared for use or used for demonstrative purposes by any witness including, but not limited to, blow-ups, enlargements of documents, x-rays, pathology slides, photographs or photomicrographs. KENTILE further reserves its right to call each witness listed in KENTILE 's list of witnesses to testify about or comment on any exhibit introduced as evidence or any items prepared for use or used for demonstrative purposes by any witness;

25. KENTILE reserves the right to call any physician or other medical practitioner who has examined plaintiff, including physicians who may have conducted independent medical examinations on plaintiff, or who may have conducted independent reviews of medical records, x-rays and pathology, as may be appropriate;

26. KENTILE reserves the right to call any and all economists or other financial experts identified by any party to this action including any economist named by any dismissed party;

27. KENTILE further reserves the right to substitute an expert witness or fact witnesses in the event the witness(es) named herein are unable to appear at trial;

28. KENTILE reserves the right to amend this list of witnesses. If other witnesses to be called at trial become known, their names, address and subject of their testimony will be reported to opposing counsel in writing as soon as they are known; this does not apply to rebuttal or impeachment witnesses;

29. KENTILE specifically notes that plaintiffs, cross-plaintiffs, and third-party plaintiffs have not identified any expert witness(es) who may testify as to theories and contentions specifically directed to KENTILE or any KENTILE product(s) at issue. If such identification is made, KENTILE may choose to amend or supplement its designation of witnesses, and may also do so in view of any designation by other parties;

30. KENTILE also reserves the right to identify later additional witnesses to testify about punitive damages, if punitive damages become an issue at trial;

31. KENTILE disclaims any duty to introduce any of the herein listed witnesses at trial by virtue of placing them on the list;

32. KENTILE reserves its right to elicit any expert opinion or lay opinion testimony at the time of trial which would be truthful, which would be of benefit to the jury to determine material issues of fact, and which would not violate an existing Court Order or the New York Rules of Civil Procedure;

33. KENTILE hereby designates, as adverse parties, potentially adverse parties, and/or as witnesses associated with adverse parties, Plaintiffs and Cross-Plaintiffs to this suit and all experts designated by any of them, even if the designation party is not a party to the suit at the time of trial;

34. In the event a present party designates an expert but then is dismissed for any reason from the suit or fails to call any designated expert, KENTILE reserves the right to designate and/or call any such experts previously designated by any party;

35. KENTILE reserves whatever additional rights it might have with regard to experts pursuant to the New York Civil Practice Law and Rules, the case law construing same, and the rulings of the trial court;

36. Subject to these express reservations, and based upon the limited information available at this time, KENTILE designates the following fact and/or expert witnesses whom it reasonably anticipates may testify, live or by prior deposition, according to availability, at the time of trial:

1. **Victor L. Roggli, M.D.**

Dr. Roggli received his medical degree from Baylor College of Medicine in 1976. He is a Board certified pathologist with a specialization in pulmonary pathology. He is a member of the International Association of Pathologists, the College of American Pathologists, the American College of Chest Physicians, and the American Thoracic Society. Since 1994, Dr. Roggli has been a Professor in the Department of Pathology at Duke University Medical Center. He is also a member of the U. S. Canadian Mesothelioma Panel. Dr. Roggli is expected to testify consistent with his study and analysis of the Plaintiffs' pathology as reflected in his reports dated July 27, 2007 and August 2, 2007 regarding plaintiff Nacht (attached) and regarding plaintiff Helfand (to be provided).

Dr. Roggli may testify that asbestos is a generic term for a group of naturally occurring fibrous minerals. Dr. Roggli may testify that there are two major groups of asbestos, serpentine and amphiboles which have different physical forms and clearance rates after deposition in human lungs. Dr. Roggli may testify that the amphibole group contains several forms of asbestos including crocidolite, amosite, tremolite, actinolite, and anthophyllite. Dr. Roggli may testify regarding the differing potential of these various forms of asbestos to cause conditions alleged by the plaintiffs, including pleural plaques, asbestosis, and mesothelioma. Dr. Roggli may testify generally regarding the role that the size, structure and chemical composition of different types of asbestos fibers play in their ability, or lack thereof, to cause conditions alleged by plaintiffs. Dr. Roggli may testify generally regarding the role of dose in determining whether certain types of asbestos fibers can cause or contribute to conditions alleged by plaintiffs. Dr. Roggli may testify generally regarding the latency periods associated with asbestos-related conditions alleged by plaintiffs.

Specifically, Dr. Roggli is expected to testify that amphibole asbestos fibers are more carcinogenic and more potent than chrysotile asbestos fibers. Crocidolite asbestos is the most potent fiber type. Long, thin fibers associated with amphibole asbestos pose the greatest hazard and that short chrysotile fibers are non-pathogenic. The difference in potency among the fiber

types and sizes is related to the low pulmonary retention of chrysotile and of short fibers. Chrysotile tends to break down in the lungs more readily and be removed as compared to amphibole fibers, which are retained and remain in the lungs for longer periods of time. Dr. Roggli will also explain the concept of respirability, and opine that non-respirable fibers do not cause or contribute to the development of disease.

Dr. Roggli will also testify regarding other potential causes of mesothelioma, including radiation. He will discuss the therapeutic radiation used to treat plaintiff Helfand for prior cancers, and will opine regarding the known proclivity of radiation to cause mesothelioma and other solid tumors. Dr. Roggli shall further testify based upon the testimony and evidence that is introduced upon the trial of all of the actions consolidated for joint trial.

2. **Charles Redinger, Ph.D., CIH**

Dr. Redinger is a certified industrial hygienist with a Ph.D. in Industrial Health from the University of Michigan. He is a Principal at Redinger & Associates, a technical consulting firm for environmental health and safety management. He has reviewed plaintiffs' testimony as well as the testimony of non-party witnesses along with documents provided by plaintiff and expert reports from plaintiff and defendants. He is expected to testify about industrial hygiene issues related to direct and bystander exposure to the installation, maintenance and removal of asbestos-containing floor tile products as well as from the use of numerous other materials and products that contained asbestos as reflected in reports dated August 22, 2007 pertaining to the Nacht and Helfand cases (attached).

Dr. Redinger has reviewed plaintiffs' occupational history and alleged exposure to asbestos for the purpose of offering his opinions regarding plaintiffs' exposure to respirable asbestos over a period of time, if any. He will discuss the different types of asbestos fiber and testify regarding the types of asbestos fiber found in the products to which plaintiffs' were allegedly exposed, and explain how different products required fibers of different types and sizes and how the different characteristics of these different types and sizes of fibers affected the dose of respirable asbestos to which each plaintiff may have been exposed. He may rely in this respect upon his review of testimony and evidence, the reports of plaintiffs' and defendants' experts regarding plaintiffs' alleged exposure to asbestos, published and unpublished studies as well as his experience, training and study in the field of Industrial Hygiene. He will testify that the proper use, installation, maintenance of removal of vinyl asbestos floor tile does not produce dangerous levels of respirable asbestos because floor tile is non friable and the fibers are encapsulated. He will opine that asbestos-containing floor tile products are not inherently dangerous. He may also critique the work and testimony of plaintiffs' experts, and may testify based upon evidence introduced upon the trial of these consolidated actions.

3. **Howard M. Sandler, M.D.**

Dr. Sandler is a licensed physician specializing in occupational and environmental medicine. Dr. Sandler is President of Sandler Occupational Medicine Associates, Inc., Co-Chairman Committee on Medical Standards: Disability and Fitness-for-Duty Section, American College of Occupational and Environmental Medicine and a member of the American Industrial Hygiene Association Indoor Environmental Quality Task Force. Dr. Sandler is expected to

testify in his areas of expertise including occupational and asbestos disease, injury and disease causation and assessment and epidemiological study as reflected in his reports dated August 22, 2007 regarding the Nacht and Helfand cases (attached).

Dr. Sandler shall testify based on his review of plaintiffs' medical records and testimony, the testimony of non-party witnesses, documents provided by plaintiff and expert disclosure provided by plaintiffs and defendants including the reports and opinions expressed by Dr. Victor Roggli. He shall review plaintiffs' medical history and alleged exposure to asbestos and shall offer opinions regarding the diagnosis, prognosis and treatment of each plaintiff including but not limited to pre-existing medical conditions and injuries. He will testify regarding plaintiffs' work-life expectancy in light of their pre-existing conditions.

Dr. Sandler will discuss the different types of fibers that are classified as asbestos, and shall explain the differences between chrysotile and amphibole fibers such as crocidolite, tremolite and actinolite. He shall explain that amphibole fibers are more carcinogenic than chrysotile fibers and how carcinogenicity is a function of factors including chemistry, shape and size. He will explain the human respiratory system and how certain fibers are respirable while others are not. He will further testify how the respiratory system attempts to clear fibers and why it is able to clear certain types and sizes of fibers more easily than others. He will describe the body's reaction to asbestos in the lung. Dr. Sandler will review the scientific literature regarding asbestos and associated diseases and will give the opinions outlined in his reports. He shall also review the scientific literature regarding exposure of those who work with or around asbestos floor tile and shall give the opinions outlined in his reports in this regard as well.

Dr. Sandler shall also testify regarding causes of mesothelioma other than asbestos, including but limited to radiation. He shall review plaintiff Helfand's medical history with emphasis on his history of cancer and the use of therapeutic radiation to treat it. He shall review the scientific, epidemiological and medical literature documenting the incidence of solid tumor cancer and mesothelioma as a result of exposure to radition and shall opine regarding this cause and effect relationship in the Helfand case. Dr. Sandler will further testify based upon the evidence introduced upon the trial of these consolidated actions.

4. **Fred Goldman, Ph.D.**

Dr. Goldman holds a Ph.D. in Economics from the City University of New York Graduate School. He is Associate Professor Emeritus of Economics at the Robert J. Milano Graduate School of Management and Urban Policy at New School University, and served as Chairman of the Graduate Program in Health Services Administration and Gerontological Services Administration at that institution. He is also a Research Associate with the National Bureau of Economic Research. Dr. Goldman may testify regarding plaintiffs' claims of economic loss, including but not limited to the extent and nature of loss of earnings or earnings capacity, loss of expected profits or business opportunity, loss of household services, past and future medical expenses, and any and all wrongful death damages allegedly sustained by plaintiffs' estate. He will base his opinions and testimony upon his review of plaintiff's testimony, employment records, payroll records, tax records, social security records, pension records, financial statements and any other records that bear on the losses alleged by plaintiffs or decedents' estate that are made available for his review. He shall further testify based upon his review of any report of alleged economic loss submitted by plaintiff and upon the testimony

given by any economist at trial by any plaintiff and the evidence presented at trial. Kentile also reserves the right to call Dr. Goldman to testify at trial to rebut the method of calculation of economic loss as testified to by plaintiffs' economist at trial.

5. **Rocco Sergi**

Former Kentile employee by deposition.

6. **Boniface T. Ramundo**

Former Kentile employee by deposition.

KENTILE reserves its rights to revise and/or supplement this disclosure response.

Dated: New York, New York
August 22, 2007

Yours, etc.

HARRIS BEACH PLLC
Attorneys for Defendant
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100 Wall Street, 23rd Floor
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(212) 687-0100

By: Andrew J. Orenstein, Esq.

To: WEITZ & LUXENBERG, PC.
Attorneys for Plaintiffs
180 Maiden Lane
New York, New York 10013

AFFIDAVIT OF SERVICE BY MAIL

STATE OF NEW YORK)
) SS.:
COUNTY OF NEW YORK)

The undersigned being duly sworn, deposes and says that she is not a party to this action, is over the age of 18 and lives in the County of Kings. On the 24th day of August, 2007 she served the within **AMENDED DESIGNATION OF EXPERT AND TRIAL WITNESSES** Upon:

JOSEPH WILLIAMS, ESQ.
WEITZ & LUXENBERG
180 MAIDEN LANE
NEW YORK, NEW YORK 10038

ALL DEFENDANTS ON ATTACHED RIDER

by mailing the same in a sealed envelope, with postage prepaid thereon, in a post office or official depository of the United States Postal Service within the State of New York.

Rhonda G. Pitt

Sworn to before me this
24th day of August, 2007

Notary Public

ELISA FRAGHER
Notary Public; State of New York
No. 01FR6019671
Qualified in Richmond County
Certificate Filed in New York County
Commission Expires February 16, 20 11

NACHT SERVICE RIDER

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#243050

AFFIDAVIT OF SERVICE BY MAIL

STATE OF NEW YORK)
) SS.:
COUNTY OF NEW YORK)

The undersigned being duly sworn, deposes and says that she is not a party to this action, is over the age of 18 and lives in the County of Kings. On the 24th day of August, 2007 she served the within **AMENDED DESIGNATION OF EXPERT AND TRIAL WITNESSES** Upon:

JOSEPH WILLIAMS, ESQ.
WEITZ & LUXENBERG
180 MAIDEN LANE
NEW YORK, NEW YORK 10038

ALL DEFENDANTS ON ATTACHED RIDER

by mailing the same in a sealed envelope, with postage prepaid thereon, in a post office or official depository of the United States Postal Service within the State of New York.

Rhonda G. Pitt

Sworn to before me this
24th day of August, 2007

Notary Public

ELISA FRAGHER
Notary Public, State of New York
No. 01FR6019671
Qualified in Richmond County
Certificate Filed in New York County
Commission Expires February 16, 20__

HARVEY HELFAND REMAINING DEFENDANTS LIST

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#243008

EXHIBIT B

SOMA
A Healthier World. A Safer Workplace.

August 21, 2007

VIA ELECTRONIC MAIL

Andrew Orenstein, Esq.
Harris Beach, PLLC
100 Wall Street, 23rd Floor
New York, NY 10005

RE: Harvey Helfand

Dear Mr. Orenstein:

Thank you for referring this case for medical review and analysis. The records indicate that this case involves a claim of malignant mesothelioma. Specifically, it is claimed that Mr. Helfand was exposed to asbestos from various sources while working doing home remodeling as well as while growing up from certain activities in his home.

I am a licensed physician specializing in occupational and environmental medicine. I was trained in the field while serving as a medical officer with the National Institute for Occupational Safety & Health ("NIOSH"), an institute within the Centers for Disease Control and Prevention ("CDC") of the United States Public Health Service. I also have trained with noted experts in this field. Occupational and environmental medicine is a sub-specialty of preventive medicine which deals with the recognition, assessment, control and prevention of potentially hazardous workplace and community exposures. I have practiced in the field since 1979. As part of my experience and training in this field, I utilize the principals and practices of toxicology, epidemiology, industrial hygiene and occupational and environmental medicine, among other related disciplines. I have consulted for NIOSH, the U.S. Occupational Safety & Health Administration ("OSHA"), the U.S. Environmental Protection Agency ("EPA"), the U.S. Consumer Product's Safety Commission (CPSC), and other state and federal government agencies. I was invited to serve on a National Academy of Sciences panel addressing causation in an occupational health concern. I also have been invited to serve on a committee within the NIOSH National Occupational Research Agenda. I have provided expert testimony on occupational and environmental health issues on several occasions before the U.S. Congress, before various state legislatures, and in state and federal court proceedings. I have taught and lectured widely in the field, including courses and lectures on causation determination. I have published and contributed to texts in the field. I also have contributed to various governmental reports and criteria for regulatory development. I currently am conducting a multi-year

epidemiological study of thousands of workers potentially exposed to various inorganic dusts including silica and asbestiform minerals. Throughout my career I have performed many assessments of possible pneumoconioses and malignancies and potential exposure in individuals and groups.

Specifically with regard to asbestos, I performed a health assessment of asbestos and asbestos substitutes for the CPSC. I conducted an epidemiologic study on a wide range of job duties at a facility where asbestos-containing materials were employed including insulation materials. I have designed and conducted medical monitoring and surveillance for various employers with asbestos-exposed workers. I have routinely evaluated and continue to evaluate the scientific and medical literature regarding exposure and health effects of asbestos and other inorganic dusts. I also have participated in the exposure analysis of various asbestos-containing products, materials and environments along with my industrial hygiene staff.

Opinions offered regarding presence of alleged disease, exposure to possible risk factors, causation, alternative etiologies, warnings and scientific state of the art regarding the development of mesothelioma and asbestos exposure are based on my training, experience, study and continuing assessment of the relevant scientific and medical literature. My opinions and conclusions are also based on my review and evaluation of the various case materials including Memorial Hospital for Cancer and Allied Diseases, Dr. Gordon Magrill, Dr. David Pfister and Dr. Craig Margulies, as well as deposition transcripts of Mr. Helfand.

Background

Mr. Helfand, born November 8, 1935 worked primarily as a printer although he did perform considerable work in primarily residential home remodeling. He testified that he smoked beginning age 15 until approximately the late 1970's – early 1980's, although the medical records indicate that he quit in 1991. One of the brands he smoked was Kent cigarettes.

Chronological Medical History

11/8/1935: Date of birth.

9/28/1984: Memorial Hospital. General anesthesia. Neurofibroma, posterior base of left neck. Wide excision of tumor, posterior left base of neck.

10/16/1984: Memorial Hospital, discharge summary. Admission 9/27 – 10/1/84. This is the first admission for this 48 year old white male who became aware of a mass arising just above the left scapula approximately two months prior to admission. The presence of the mass was associated with no local pain or discomfort and there was no evidence of local infection nor of any recent history of trauma. Patient underwent incisional biopsy of the mass with findings of neurofibroma with atypical. Patient remains essentially asymptomatic but he is still aware of the persistent mass beneath the biopsy site. His general health has been good except for seasonal allergies to certain pollens. He did smoke 2½ packs of cigarettes daily but completely stopped two years prior to admission. Chest normal.

Summary of management – Wide excision of the left posterior neck mass was performed without complication. Pathology showed diffuse atypical neurofibroma with focal low grade malignant change infiltrating skeletal muscle. Negative skin with scar and foreign body giant cell reaction. Postoperative evaluation found the patient in stable condition and an uneventful hospital course ensued.

5/22/1986: Memorial Sloan Kettering Cancer Center, radiation oncology. 51-year-old printer, married with three children. Smoked until three years ago and was generally well until September 1984. At that stage he presented with a small lesion in his left trapezius muscle. Patient was taken for operation and this tumor was removed locally. Histology was neurofibroma with atypia. Followed up closely but remained well until December of last year when he started to feel local recurrence near the previous excision area. This grew until he required a local excision once more. Patient returned to Dr. Strong for a radical excision of the previous scar and the muscles was carried out. The histology this time was similar, however, because of the previous history of local recurrent it is decided to send him for radiation therapy. Two lymphnodes biopsied were negative at levels II and III on frozen section. Patient probably requires postoperatively radiation therapy.

6/4/1986: Gordon S. Magrill, M.D., Memorial Hospital. Discussed the pros and cons, pointed out chemo and radiation would hopefully cut the change of recurrence from 80-90% down to perhaps 25-35%. He will discuss this with the patient.

8/7/1986: Memorial Hospital, diagnostic radiology. Chest. In comparison with the study of 5/13/86 there has been no interval change, again demonstrating no active disease.

8/23/1986: Memorial Sloan Kettering Cancer Center. Developed small lesion in his left trapezius muscle in 9/84. Local excision revealed a neurofibroma with atypia. Patient developed recurrent disease in the local excision site in 12/85 and had a second excision. He then had a radical excision of the previous scar and surrounding muscles. The patient then received an alternative course of chemotherapy and radiation therapy. Treatments were well tolerated with some skin erythema in the treatment field.

Left trapezius muscle, 1.25 MeV, 1400; Left trapezius muscle, 1.25 eV, 1500; Left trapezius muscle, 1.25 eV, 1500.

12/11/1986: Memorial Hospital, diagnostic radiology. PA & lateral chest. The heart is within normal limits in size and configuration. A questionable small vague density is seen projected over the right mid-lung field and the anterior right third-rib interspace which cannot be seen definitely on the lateral film or on the prior exam of 9/11/1986. A repeat chest PA with bilateral 10 degree oblique projections is suggested to determine if this represents a small pulmonary nodule or superimposed normal structures.

1/29/1987: Memorial Hospital. Solid tumor service. Patient SP resection of a neurofibrosarcoma of the left neck with positive margins and some high grade areas who was on

combo chemotherapy and radiation therapy from June to December 1986. Last treatment was on 12/11/86. Dr. Strong satisfied with local area. Patient denies any new problems.

1/29/1987: Memorial Sloan Kettering Cancer Center. PA and lateral chest. The lung fields are clear. A nodule questioned in the right mid right lung field is not seen on today's examination. Deviation of the trachea to the left persists, unchanged and suggests a right thyroid mass. The chest is otherwise normal.

2/26/1987: Memorial Sloan Kettering Cancer Center. Chest, PA and lateral. There has been no change in the chest x-ray since 1/29/1987. The lungs appear clear. The heart size, hilar and mediastinal contours appear normal. The trachea is displaced in the neck suggesting the possibility of an enlarged right lobe of the thyroid gland.

4/10/1987: Memorial Sloan Kettering Cancer Center. Chest. No nodules or other abnormalities are detected. Please note that tracheal deviation is not optimally evaluated on oblique projections.

1/7/1988: Memorial Sloan Kettering Cancer Center. Chest. No active disease or interval change as compared to 9/8/87.

6/6/1989: Memorial Sloan Kettering Cancer Center. PA and lateral chest. In comparison with the study of March 7, 1989 there is an infiltrative process approximately 3 cm in diameter along the lower right lateral chest wall which has more the appearance of an inflammatory process and in view of the recent history of cough and malaise appropriate anti-inflammatory is recommended before considering the diagnosis of metastatic disease on follow up films. No other change.

6/13/1989: Memorial Sloan Kettering Cancer Center. Heart is of normal size and configuration. There are vague infiltrates suspected at the periphery of the right lower lung field on the PA projection, though these cannot be clearly seen on the lateral film. These appear slightly less than on 6/6/89 and clinical correlation is suggested.

6/27/1989: Memorial Hospital. 53-year-old gentleman with neurofibrosarcoma resected from left neck three years. The patient developed a right lower lobe infiltrates 6/6/89 treated with three weeks of antibiotics which he stopped yesterday. The infiltrate improved. CXR shows almost complete clearing of the infiltrate with sequential resolution of the density. The patient feels well. He has a KPS of 80%. He resumed working.

7/27/1989: Memorial Hospital. Follow up visit. History or neurofibrosarcoma resected times two. Second resection followed by radiation therapy and chemotherapy. Has flu type symptoms with an infiltrate in 6/89. Chest x-ray today shows no evidence of the infiltrate initially documented 6/6/89.

7/27/1989: Memorial Sloan Kettering Cancer Center. PA and lateral chest. The patchy infiltrate present in the lower portion of the right lower lobe has resolved. The lungs are clear. There is no evidence of adenopathy or pleural effusion.

7/11/1991: Memorial Hospital. Lungs are clear. Heart sounds are normal. Head and neck exam shows no palpable nodes on his right side, his left side shows a clean scar with normal post surgical changes. Chest x-ray continues to be NED with no infiltrates or nodules.

7/11/1991: Memorial Sloan Kettering Cancer Center. Sarcoma, interval change. There is no evidence of pulmonary infiltrate or pleural effusion. The cardiac size is normal and the mediastinal silhouette is otherwise unremarkable.

2/19/1992: Memorial Sloan Kettering Cancer Center. PA and lateral chest. Sarcoma. Current exam does not show any lung infiltrations. Some prominent markings in the right lower lung. Costophrenic angles are clear. Heart within normal limits.

10/17/1992: Memorial Sloan Kettering Cancer Center. PA/Lateral chest. Clinical history: sarcoma of the chest. Current exam does not show any significant interval change compared to previous exam. There are no new developments.

1/14/1993: David Pfister, M.D. Memorial Hospital, out patient progress record. Follow up consultation. He is here for routine follow up after his treatment of sarcoma. No pathology or adenopathy in neck. Primary site in left neck clean. Performance status 90%. Weight 89 kg. BP 130/90, temp 35.1. Regular cardiac rhythm. Bilateral breath sounds. Abdomen, soft non-tender. Continues to be NED. Chest x-ray was reviewed and revealed no evidence of disease.

1/9/1995: Memorial Sloan Kettering Cancer Center. Bladder cancer. Specimens submitted-bladder tumor; base of bladder tumor; prostatic urethra. Bladder biopsy – papillary transitional cell carcinoma with extensive squamous metaplasia, grade III/III. No muscularis propria is identified. Bladder, base biopsy – papillary transitional cell carcinoma with extensive squamous metaplasia, grade III/III. No muscularis propria invasion is identified. Bladder, prostatic urethra, biopsy – transitional cell carcinoma in-situ.

3/21/1995: Memorial Hospital for Cancer and Allied Diseases, discharge summary. Admission diagnosis: Transitional cell carcinoma of urinary bladder. History of recurrent neurofibrosarcoma of left neck.

During workup for hematuria, the bladder tumor was discovered and resected, pathology revealed papillary transitional cell carcinoma of urinary bladder and transitional cell carcinoma in situ of prostatic urethra. He is now admitted for interval cytoscopy/TURB. History of recurrent neurofibrosarcoma of left neck, status post resection.

8/31/2006: Craig Margulies, M.D., Digestive Disease Center. Pelvis was normal, but the abdomen CAT scan showed left lower lobe nodule of the lung associated with left pleural effusion, and a dedicated CT scan of the chest was recommended. Mild emphysematous

changes, vague pulmonary nodules, which after consultation with two cardiothoracic surgeons, was thought to be due to scarring from previous lung infections. Also had left lung base nodule, small pleural effusion, asymmetric muscle hypertrophy on the left upper back, which probably represents trapezius muscle surgery in the past. Developed increasing fluid in the left lung. This ultimately resulted in a throacentesis, and this was performed on 8/29/06. The results of the cytology revealed lymphocytes and diffuse small clusters of atypical mesothelial cells. Pathology recommended repeat cytologic examination if the fluid reaccumulated. There was no definitive diagnosis of cancer.

10/3/2006: Memorial Hospital for Cancer and Allied Diseases, record of operation. Recurrent left pleural effusion, with metastatic disease or primary tumor involving the pleura. Flexible bronchoscopy, left open pleural biopsy.

10/3/2006: Memorial Hospital for Cancer and Allied Diseases, surgical pathology report. 70-year-old male with history of left neck liposarcoma, h/o bladder cancer and bilateral pulmonary nodules. Pleura, left biopsy – malignant mesothelioma, epithelioid type. Portion of rib, left, sixth excision – fragments of benign bone and normal hematopoietic bone marrow. The immunohistochemical stains show the tumor cells to be positive for calretinin, WT1 and AE1:AE3. While they are negative for TTF-1, CEA, CD15, B72.3 and BEREP4. Mucin stain is negative.

10/11/2006: Memorial Hospital for Cancer and Allied Diseases, outpatient progress record. 70-year-old man, former smoker and history of hypertension, GERD, pancreatitis, kidney stones, angioplasty in 1998 and also with liposarcoma of the neck diagnosed in 1983, status post surgical resection and chemotherapy and localized radiation therapy. Also has a history of transitional cell carcinoma of the bladder that was treated with a transurethral resection of bladder tumor in 1993 and 1995.

Long-standing back pain that he describes as sciatica. Worsened in the beginning of year to point he had surgery for disk herniation on 5/11/06. During workup for that surgery, chest x-ray was abnormal and subsequently led to a CT of the chest. This showed focal pleural thickening in the right lung with lymphadenopathy in the right hilum and mediastinum, as well as bilateral axillary lymph nodes. Dr. Rusch found that he had some indeterminant pulmonary nodules but there was nothing significant to biopsy and that she recommended observation with a repeat CT scan in six months.

Because he was having symptoms of increasing shortness of breath, he had repeat CT scan that showed a new left-sided pleural effusion. At that point, he had a diagnostic thoracentesis at an outside hospital where by report the cytology was negative. Symptoms improved mildly over the next month but he still had increasing shortness of breath. Because of new symptoms and CT scan that showed a new pleural effusion, he scheduled an appointment to see Dr. Rusch for an inpatient admission for drainage of the effusion and biopsy of his pleural based mass.

Underwent PET scan on 9/13/06. Findings showed a large amount of loculated fluid throughout the left hemithorax with compression atelectasis of portions of the left lung. Some hypermetabolic activity in the left lung. There were some areas of pleural thickening noted especially in the medial aspect of the left hemithorax and the region of the left costophrenic sulcus that were noted to be hypermetabolic. Repeat chest CT showed a large left-sided pleural effusion. Difficult to tell if there was an underlying mass or pleural thickening on that CT scan.

He notes about a 15 pound weight loss over the last few months. Still complains of some shortness of breath especially on exertion but has no chest pain or fevers. He does note an intermittent dry cough but denies wheezing. The remainder is unremarkable in detail.

He is married with three daughters. Worked at a printing company for many years and was exposed to chemicals on a daily basis. Positive tobacco history with about 20 pack-years but quit 15 years ago. Mother and father died of heart related events.

Surgical pathology has not been finalized at this point. The surgical notes from the biopsy show that a frozen section result was malignant epithelioid neoplasm. After speaking with the pathology resident, and Dr. Travis, it is their feeling that this is most consistent with mesothelioma. The initial immunohistochemistry is positive for calretinin. They will finalize their diagnosis in the coming days.

This is a 71-year-old man with presumed new diagnosis of a malignant mesothelioma. Long discussion with patient and wife about the natural course of this disease and that he may have been exposed to asbestos in that past and that this also could be related to his radiation therapy to the neck.

DEPOSITIONS

Harvey Helfand taken on January 22, 2007.

Mr. Helfand was born in Brooklyn on 11/8/1935. Currently resides at 19 Birmingham Lane, New Jersey. He moved into this residence in 1998. His wife's name is Leone. She was diagnosed with thyroid cancer about three years ago. Mrs. Helfand held a job as a school aide. She retired six or seven years ago. Mrs. Helfand is not and has never been a smoker.

He does not believe he was exposed to asbestos in the Birmingham Lane residence. Prior to this he lived at 1151 East 82nd Street in Brooklyn for 33 years. While waiting for the new residence they lived with a daughter for three months. The 82nd Street residence was a one-family house. He did not think he was exposed to asbestos although he did a lot of renovations such as completing a basement and putting in an extra bathroom. He indicated he "positively was exposed to asbestos there." He re-insulated the back wall of the house due to poor insulation. He recalled that he installed a vinyl asbestos Kentile floor. There was no tile there previously. He did all the work with his brother and father. He also handled the drop ceiling and stated that it would be dusty when the ceiling tiles were cut. He indicated the manufacturer of these was Celotex. He also handled the sheetrock and the cement/spackling compound. He did

not recall the manufacturer. He indicated it took a couple of years to finish the basement. He denies wearing any mask or respirator.

Mr. Helfand also installed an outdoor fence. He was not sure if the cement used contained asbestos.

He reinsulated the wall about ten years after moving in. He denies any assistance on this installation. He does think he was exposed to asbestos at this time as well due to the use of sheetrock and spackling compound. He denies using a mask or respirator at this time.

Mr. Helfand also installed a kitchen about 15 years prior to moving out. He ripped out the entire kitchen and plumbing. He hired a cabinet maker to install the cabinets. Mr. Helfand installed the floor, Amtico tiles. He handled these tiles, sheetrock and spackling compound. He denies wearing a respirator or mask when working on the kitchen renovation.

Prior to 82nd Street residence, the family lived at 1127 East 83rd Street, Brooklyn. This was a two family house that he rented. He does not believe he was exposed to asbestos at this residence although he did do some work for the landlord.

When he was 14 he moved into his family's home, which was a one family converted to a two family on Schenectady Avenue in Brooklyn. He feels that he was exposed to asbestos there due to handling Kentile floor tiles and sheetrock. He worked with his father to finish the basement and make an apartment in the basement. The work was done in the summertime and it took about six months. He also used the spackling compound. He denies using a respirator or mask during this work.

Prior to this he lived at 9802 Foster Avenue in Brooklyn. He was sure he was exposed to asbestos at this residence because he handled insulation around a boiler. They had a machine shop in the basement. He and his brother would make modifications and clean up the boiler in their basement.

Mr. Helfand has liposarcoma, which he first discovered because he felt a bump. He received surgery to remove the growth and the surrounding area. Within a year it grew back so the whole trapezius muscle was removed. He then received radiation and chemotherapy. The second surgery was done by Dr. Strong at Sloan-Kettering. He received nine cycles of chemotherapy and 30 radiation treatments twice a day. The results were that he is "still here."

He discovered he had bladder cancer after he had an infection and saw a urologist. He did not think the urologist was knowledgeable and asked his internist to perform a sonogram which revealed a spot. He was told that the bladder cancer may have been caused by the chemotherapy and partly by the inks he used as a printer.

While in high school he worked for Rabin Typographers in Manhattan for about a year. He was the clean up boy, picked up the lead, the type and cleaned the linotype machines. He had to keep the area clean and clean the lead pots, which he states were lined with asbestos. He

states he was positively exposed to asbestos at this job. He indicates there was always dust and residue all over the machines. In describing the asbestos in the pot he indicates that it was hardened already because it went on like paste. He denies wearing a mask or respirator.

He then worked for the Doughnut Corporation of America as a mail clerk.

Mr. Helfand attended a vocational high school, but he indicates that he really did not learn anything. He was not sure if he was exposed to asbestos while attending East New York Vocational School. He was in the automotive shop. General automotive work was performed there including tearing down some engine parts and brakes.

In 1953 he got a job as a helper errand boy in a lithographic shop, United Offset Company, running errands and cleaning. He requested to be on the night shift so he could learn the shop. He then went to work for Ramapo Litho Corp. His jobs were to take packages, artwork to the customers, pick up same. He would have to wash up the machines, grease and oil them, maintain them for the morning shift. He denies using a mask or respirator. He worked there for about eight years. His job title was pressman.

He also worked at Muree Press, Service Offset, Ross Printing, Triple M. H&H Multicolor was Mr. Helfand's company. Spectrum Printing was his last employer. He worked at Muree for three or four years on the day shift with his title being foreman. He prepared jobs for the shop, oversaw the different presses, quality control, production, customer relations, general supervision. He has no idea if he was exposed to asbestos at this job. He probably worked two years for Service Offset, full time on the day shift as a foreman. Same duties primarily. Some hands-on work. Again no idea if he was exposed to asbestos here. He worked six months for Ross Printing with a job title of pressman. His job duties included maintaining and operating the offset press.

Harvey Helfand taken on January 29, 2007.

Mr. Helfand worked for Triple M in Manhattan for two years as a foreman. Duties were production, quality control, scheduling, cleaning up. Full time job during the day shift. He denies wearing a mask or respirator.

He then started his own commercial printing, trade printer business, H&H Multicolor in 1970. He closed it in 1991 or 1992. After H&H he worked at Spectrum Printing until 2006. He left because he had mesothelioma and could not travel to the city. At Spectrum he was a production person where he worked in customer relations, estimating, production and deliveries.

During jobs he has taken off time to do home improvement work. He could not recall actual locations. He started this type of work when he was 15 and stopped about one year ago. He did just general contracting work, nothing major. He also did demolition and cleaning out. All of it was ripping down old stuff and modernizing. 99% was residential locations. He was exposed to pipe covering from boiler wraps and old ceiling tiles. He would rip down the old stuff as well as put the new stuff in. He would have to remove the boiler wrap which was like a

cast. Toward the middle to end of his career he would install pipe covering, but not asbestos containing covering. He thinks the wrap was white, a lightish color. He had to break it up to remove it. In regards to the ceiling tile, he would have to rip them down when there was a leak or deterioration. He mentioned some manufacturers of ceiling tile as U.S. Gypsum, National Gypsum, Kaiser, Georgia Pacific and Ready Mix. Sometimes he added powder to the Ready Mix to get it to set quicker.

He was exposed to asbestos while cutting the floor tile. He used vinyl asbestos. He would have to cut it to fit and everything was an exposure. Most of his career, Kentile was the main product he used. Congoleum and Amtico were the upgrades in tiles, sheets goods. There is also Mannington Mills.

He first heard about possible health hazards associated with asbestos about 20 years ago. Mr. Helfand was a member of Union 51, the pressman union for about five years and Local 1 of the Amalgamated Lithographers of America. He could not recall talking about asbestos in any of the union publications.

Mr. Helfand started smoking at age 15. He quit at 25-30 years ago because it was no good. He smoked about a pack a day. He smoked Marlboro but had also smoked Kent, although he did not like the taste of them. He smoked out of nervousness. He alternated between Kent and Parliament.

The manufacturer of the linotype machines was Mergenthaler.

There was a powder that he used to clean the pot. He would do the clean up after the pot was closed. Occasionally someone had to change the asbestos lining.

He worked for Service Offset for about two years, right before he started to work at Spectrum.

Mr. Helfand installed four or five floors a year: When taking up a floor he could not tell who made it. Sometimes the tile would come up in chunks other times it would come up whole.

He did a lot of residential work in Brooklyn, Queens and some on Long Island, mostly private homes. He worked mostly on basements and kitchens, sometimes refurbishing other rooms. He would resurface walls that were cracking and peeling. He indicates he installed Kentile Floors in basements, kitchens, laundry rooms and sometimes bathrooms.

Harvey Helfand taken on March 9, 2007.

According to records he worked at Spectrum in May 1993 through 2006. The presses were 2 Harris' and a Komori. He believes he was exposed to asbestos here because there was a clutching mechanism, brake system, but he could not tell for sure if it had asbestos in it.

In 1970, he worked for Lithocraft Corporation as a foreman. He indicates the mechanics of a Zenith is a very cheap operating system so he felt he was exposed to asbestos from this.

He denies ever seeing documents indicating that there were any asbestos products in Harris LTW, nor did anyone ever tell him. He worked on Harris Presses all his life so if the presses contained asbestos, then he was exposed.

In the 60's and 70's he used the powder and the ready mix joint compound. He thinks he first started using Georgia Pacific ready mix in 1953 or 1955. He would have to add water to the ready mix. He used the ready mix in mostly residential locations. He would use Georgia Pacific Ready Mix about four or five times a year. He would smooth down the ready mix, let it dry, then sandpaper it smooth. The dust would be flying all over the place. After that dried he would apply another round of ready mix with a wider knife. After that he did a third application. When he first started, he was very poor at plastering and it could take him five or six applications. Most of the plastering on these jobs he did himself. He denies wearing a mask or respirator. He did not ventilate the room.

Georgia Pacific ready mix came wet.

Based on total career, he did 70% printing and 30% home improvements. Even while working as a printer he did home improvements. He always did side jobs, he would work them nights and weekends.

He usually worked alone when working with joint compounds. He indicates that 80% of his home improvement jobs involved joint compound. On occasion he had to install light or fans and this would involve snaking electrical wire so he would have to make holes. If he made holes, he had to repair them.

He used Kentile floors primarily because of pricing. He used it on most of his jobs because of price and he had samples. He could tell the difference between vinyl asbestos and non-asbestos tile because vinyl tile is not pliable, it is very solid.

Mr. Helfand's hobby is to fly a plane. He had a friend as his mechanic. He may have occasionally helped to change brakes but the heavy stuff was done in the shop. He never changed out a clutch.

He was first exposed to asbestos pipe covering as a kid of 8 or 9 in his family home from an old boiler. The boiler was lined with white plaster and the pipes' wrapping was constantly cracking and the dust was flying. He did occasionally have to put pipe covering back on the pipe. He was told that when asbestos flakes it is at the danger point.

While employed at Rabin he did see asbestos being removed from the pot on the Mergenthaler. They would have the same white debris that had been around the boiler. They would use a mallet and screwdriver to chip it out and it would get very dusty. He believes he

breathed that dust. It was Mr. Helfand's job to clean up the asbestos removed from the Mergethaler machine.

When having to cut vinyl asbestos tile, which was not flexible, he would have to score it then snap it and get all that dust in his face. There would be a whole pile of dust, he would breathe in, cough a couple of times and move on to the next piece.

CONCLUSIONS

Presence and Extent of Alleged Injury/Illness

Mr. Helfand was diagnosed with malignant epithelioid mesothelioma in 2006 at the Memorial Hospital for Cancer and Allied Diseases in New York (Sloan Kettering). He had previously been diagnosed with and treated for neurofibrosarcoma in his upper back and neck in 1984 and a recurrence in 1986 as well as papillary transitional cell carcinoma of the bladder in 1995. He underwent localized radiation therapy along with chemotherapy from June to December 1986.

Mr. Helfand has a number of other health conditions consisting of coronary artery disease and hypertension, sarcoma, urinary bladder cancer, GERD, pancreatitis, kidney stones and lumbar degenerative disease requiring disc surgery. Had not Mr. Helfand developed mesothelioma, a diagnosis to which he will likely succumb over the next year or two, his expected life span would have been shortened by the presence of other multi-organ system disease. Additionally he would likely have been disabled to a significant extent from a number of normal life activities and his remaining years would have required other significant medical care.

Exposure

Mr. Helfand testified that he believes he was exposed to asbestos through floor tile installation and removal, using spackling compound and working around boiler and pipe insulation. He further testified that he worked 70 percent of his time in printing and 30 percent in home remodeling and that he would install 4-5 tile floors per year. The tile floors would be in kitchens, basements, and bathrooms. Kentile tiles were the most commonly used tile by Mr. Helfand. He was also potentially exposed to boiler insulation including boiler wraps and pipe insulation. This occurred as a young child in his home where he assisted his father with the boiler in the basement as well as during the performance of home remodeling. He also reportedly installed and removed a Kentile floor from one of his residences. Finally he reported having to snake wires while performing electrical work during home remodeling activities over many years.

Causal Association

It is my opinion that Mr. Helfand's unfortunate mesothelioma was not the result of work with or in and around asbestos-containing floor materials. It is my further opinion that his tumor

if indeed the tumor is asbestos related at all, is the result of exposure to amphiboles from other non-flooring related, asbestos exposures, likely amphiboles from the work around boiler insulation or other insulation. Insulation in the United States contained substantial levels of the amphibole amosite, and amosite is the most commonly found fiber type in the lungs of those with mesothelioma (Roggli et al, 2002; Dodson et al, 2005). It may also be the result of familial (heredity) factors as will be discussed below. However, as discussed below, his mesothelioma was also likely a result of exposure to therapeutic radiation required for his 1986 of a back/neck sarcoma.

Over twenty studies (see references) of removal of existing asbestos-containing floor covering using recommended procedures as well as installation and use of such floor covering have shown from the 1970's through the 2000's that exposures to floor tile work such as in the case of Mr. Helfand has not been found to produce exposures in the range where increased risk of asbestos-related disease has been identified including for mesothelioma. Generally their eight (8) hour, time weighted average exposures are in the range similar to that of brake/auto mechanics, at 0.02 – 0.002 f/cc. Further it is noted that Mr. Helfand said that he only did four to five floors per year which would severely limit the fiber year cumulative dose that Mr. Helfand would have received from that particular potential asbestos exposure source.

As knowledge of the potential for exposure to airborne asbestos fibers from work with asbestos-containing materials such as braking material and vinyl asbestos tile began to be published in the 1970's and concern for health risk at lower levels of exposure than previously thought grew during that same time frame and beyond, various studies have been performed which shed considerable light on the level of risk for the development of mesothelioma among workers performing these activities. Information concerning mesothelioma risk comes from three scientific pursuits:

- Epidemiological studies of workers performing brake repair and replacement and other activities, e.g., grinding and drilling.
- Lung tissue fiber burden analysis of various "exposed" and "unexposed" workers and general population individuals with mesothelioma.
- Formal risk assessments using epidemiological studies to predict risk of developing mesothelioma at low levels of exposure.

Key factors surrounding issues of potential for mesothelioma development secondary to work with asbestos-containing floor covering center around the following:

- Chrysotile asbestos was used virtually exclusively in the production of floor covering materials.
- Epidemiologic studies of "pure" chrysotile study populations (cohorts) do not show elevated rates of mesothelioma for such populations.

- Scientific evidence from the study of chrysotile miners/millers indicate that risk of mesothelioma in so-called "pure" chrysotile-exposed populations is likely related to exposure to the contaminant amphibole fiber, tremolite, in those operations.

- Although not totally removed from the milled chrysotile product used to manufacture the asbestos-containing floor covering, fiber burden analysis of brake workers with similar workplace exposures to floor mechanics shows similar levels of tremolite in workers as compared to members of the general population indicating that the removal was sufficient to avoid appreciable exposure to tremolite fibers to those working with chrysotile-containing products.

- Studies of fiber type/burden in patients with mesothelioma virtually always show the presence of amphibole fibers, primarily amosite, tremolite and crocidolite.

- Actual epidemiological studies of brake workers who again have similar asbestos exposures to floor mechanics do not show an increased risk for the development of mesothelioma.

- Although the exact level of what can be termed "non-asbestos related mesothelioma" is debated, at least ten to twenty percent of mesotheliomas are not the product of asbestos exposure.

Seven epidemiological studies, all case-control studies, provide no scientific evidence for an increased risk of mesothelioma in auto mechanics exposed to asbestos from brake and clutch work. The relative risk or odds ratios and corresponding 95% confidence intervals for each study along with the number of mesothelioma cases and dates of diagnosis are provided chronologically:

McDonald and McDonald (1980) – 480 cases in Canada (1960 – 1972) and United States (1960) – RR/OR – 0.91 (0.39 – 2.13)

Teta et al (1983) – 220 cases in Connecticut (1955 – 1977) – RR/OR – 0.65 (0.08 – 5.53)

Spirtas et al (1985, 1994 update) – 259 cases in NY and LA (1975 – 1980)
RR/OR – 1.00 (0.60 – 1.60)

Woitowitz and Rodelsperger (1994) – 324 cases in Germany with motor mechanics beginning employment 1925 – 1972 – RR/OR – 0.87 (0.46 – 1.64)

Teschke et al (1997) – 51 cases from British Columbia (9/90 – 8/92) –
RR/OR – 0.80 (0.20 – 2.30)

Agudo et al (2000) – 132 cases in Spain (1993 – 1996) – RR/OR - 0.62 (0.17 – 2.25)

Hessel et al (2004) – expanded Spirtas studies of 1985 and 1994 –
RR/OR – 0.71 (0.30 – 1.60)

A meta-analysis of these case-control studies by Otto Wong published in 2001 reported a relative risk for all studies combined of 0.90 (0.66 – 1.23). It should be noted that the relative risk value for the Agudo study was calculated by Dr. Wong. The Goodman et al, 2004 meta-analysis also noted no increased risk for mesothelioma. A recent article by Lemen in 2004 calls these findings into question; however, there are numerous deficiencies in his approach limiting the applicability of his opinions. Harvard researchers, Laden et al also in 2004 performed a critical review of all automobile mechanics epidemiological studies and stated "...evidence did not support an increase in risk of either lung cancer or mesothelioma among male automobile mechanics occupationally exposed to asbestos from brake repair." Finally, Butnor et al in 2003 reported on fiber lung burden in 10 cases of brake-dust exposed individuals. Although the exposure for the significant amphibole presence was not reported, a frequent problem in asbestos studies, all ten have excess commercial amphibole fibers in their lungs.

There is consistency among all of the studies that report a relative risk of one (1) and usually considerably less. Relative risk of one or less indicates absence of increased risk although one cannot say that there is a deficit of risk as the confidence intervals does not provide statistical significance. It should be stressed that the time frames of exposure occurred primarily when compressed air for "blowing" out brake dust was used. As noted previously, this would correspond to the potential for highest asbestos exposure. Additionally, the studies examine populations through North American and two European countries providing further evidence of consistency in different study populations and conditions. While earlier case reports or series (Woitowitz and Rodelsperger, 1991; Langer and McCaughey, 1982; Huncharek et al, 1989) have reported limited findings warranting further study, such study (i.e., case-control publications discussed above) has not shown an association. Case reports/series are hypothesis-generating in nature and especially when compared against more formal epidemiological investigations that do not have the ability in and of themselves to demonstrate association or causation. It is expected that some cases of mesothelioma will occur simply due to chance. The three case reports by Dr. Grace Ziem referenced in Dr. Castleman's textbook and cited in the 1996 EPA guidance document are interesting, but they have not been reported in the open literature and it is unclear if other exposures were potentially operative, if the diagnosis was confirmed and other issues of causation were satisfied.

Studies exploring fiber type and burden in individuals with mesothelioma have been performed by a number of investigators. These studies are useful in exploring the presence of amphibole fibers versus chrysotile fibers. However, such studies while consistently demonstrating exposure to amphiboles, dose response data comparing amphibole burden versus chrysotile burden cannot be confidently generated due to ability to eliminate chrysotile fibers preferentially over amphibole fibers as amphibole fibers are much more likely to be retained in lung tissue over the years. Nonetheless, such studies do call into question, those opinions which attempt to relate mesothelioma to chrysotile exposure especially at extremely low exposure levels such as that which has been demonstrated to occur in brake repair and replacement workers (see exposure section above). Beginning in the early 1980's research by McDonald in

Canada and an assessment by Churg in 1982 along with substantive studies by Churg, Roggli and others, yielded information regarding the fiber types and levels of fibers in the lungs of patients with mesothelioma. Such studies showed that amphiboles were virtually always present and in appreciable levels especially in comparison to chrysotile fibers. Churg in 1982 stated:

> "Analysis of pulmonary fiber burden suggests that asbestos-related disease is not merely a matter of total number of fibers represent, but that factors such as fiber type and size are equally important."

Further commenting mesotheliomas, Churg stated:

> "These observations suggest that most mesotheliomas are associated with numbers of increased numbers of commercial amphiboles and not with chrysotile asbestos."

Churg et al in 1984 performed a fiber analysis of lung asbestos content in five chrysotile (Quebec) workers with mesothelioma. This study of six (6) patients found both tremolite and chrysotile in the lung tissue however, the tremolite levels were 9.3 times the controls and concluded that tremolite exposure/content may be important in the pathogenesis of these tumors.

McDonald et al in 1989 reported on 78 Canadian mesotheliomas diagnosed 1980 – 1984 attributing approximately 68 percent of the Canadian tumors to amphibole fibers, especially tremolite, and virtually no mesotheliomas to chrysotile fibers. Churg and Wright also in 1989 evaluated nine matched pairs of mesothelioma patients who were either shipyard and insulation workers, or chrysotile-industry workers. The researchers report that a relatively huge chrysotile content would be necessary to induce mesothelioma on the order of three times that necessary for asbestosis development. However, the amphibole level associated with mesothelioma was 1/35 that seen in amphibole-related asbestosis indicating a potential 100 fold difference between the ability of amphiboles and chrysotile to produce mesothelioma. Gibbs et al in 1990 reported on 10 paraoccupational cases of mesothelioma patients in England whose husbands had worked with asbestos. The fiber analysis showed only one person with increased chrysotile burden however that individual also had an increased level of amphibole fiber as well. Two individuals had "normal" levels of both amphibole and chrysotile fiber types. Rogers et al in 1991 reported on 221 mesothelioma cases in Australia and found only two cases with "chrysotile only" fibers in the lung tissue with the authors concluding that the greatest risk for mesothelioma in Australia was exposure to crocidolite, especially those fibers greater than 10 um. Interestingly, there was only a significant dose response effect for short chrysotile fibers which is not consistent with regulatory or risk assessment evaluations (see Risk Assessment below). Churg et al in 1993 reported on 19 chrysotile miners/millers with mesotheliomas noting that the mesothelioma burden is probably tremolite-related and that the role of chrysotile remains uncertain. Sakai et al in 1994 reported on 16 mesothelioma patients gathered over 1983 to 1990 from 15 Japanese hospitals. All 16 have significant pulmonary fiber levels of amosite and/or crocidolite.

A 1995 study by Dufresne of 12 Canadian chrysotile asbestos miners with malignant mesotheliomas. Two of the 12 cases were felt to be attributable to important crocidolite and amosite fiber, while the other ten where associated primarily tremolite or a tremolite/chrysotile

combination. All twelve had tremolite fiber present. In 38 mesothelioma cases in Canadian asbestos miners, millers and factory workers from the Thetford and Asbestos operations, McDonald et al reported in 1997 that all had tremolite in the lung tissue except two; however, those had crocidolite. The authors noted that the predominance of the cases were from the Thetford mines and those workers had generally worked in specific areas where tremolite presence was considerable. Some workers had also been exposed to crocidolite and amosite fibers, apparently used in factories for production of gas masks. The enhanced retention of tremolite fibers from the lungs of workers in the Thetford mines was confirmed by Nayebzadeh et al in 2001.

Some studies have reported that mesotheliomas are present in individuals with chrysotile only in the lung tissue. Nolan et al in 1994, reported that in one case of mesothelioma with high levels of chrysotile in the lung tissue that amosite and crocidolite fibers were not found. The authors specifically conclude that they found no evidence of "commercial amphiboles." However, the authors report that almost one (1) percent of the fibers were silicate fibers also known as the contaminant tremolite. In 2002, Suzuki and Yuen reported on the fiber types and dimensions found in the lungs and mesothelioma tissue of 168 patients with mesothelioma. These patients had a variety of exposures to asbestos although specific associations with exposure history were not reported. While some patients had chrysotile only in their lung tissue, the majority had chrysotile only in their mesothelial tissue. It is critical to note that the researchers included very small fibers, i.e., shorter than one micron in length. Such fibers have not been implicated in the production of asbestos-related disease including mesothelioma. The mere presence of short chrysotile fiber in tumor tissue does not indicate causation, merely that fibers of that dimension can gain entry to tissues outside the lung. Additionally for the 11 patients with chrysotile only in their lung tissue, the fiber size is not identified. This study has received considerable criticism in the scientific arena. Thus, an association between fibers of the size considered to be pathogenic was not indicated for exposure to chrysotile fibers alone.

Roggli et al reported in 2002 on 1445 mesothelioma cases and fiber type and burden in 268 of those cases in correlation to their occupational and environmental exposure to asbestos. As in other studies of this study population type, the predominate fiber found was amosite. No cases were reported as having chrysotile alone. Importantly, the fiber levels of amphiboles, especially noncommercial amphiboles, and chrysotile found in lung tissue were similar for workers in the automotive as those whose only possible exposure to asbestos was having been building occupants. The eleven automotive brake repair workers in the automotive category had "either an elevated amosite content or an asbestos concentration indistinguishable from background," indicating that such workers who developed mesothelioma did not receive exposures and therefore were not at increased risk over "unexposed" populations. The authors noted "brake dust is unlikely to cause mesothelioma." Dodson et al, in 2003 reported on asbestos burden and fiber type in 15 women with mesothelioma who had worked in various occupations. The most commonly found fiber was amosite and the next most common was tremolite. Chrysotile was not reportedly found in 11 cases and there were no cases of chrysotile fiber found alone. Butnor et al, in 2003 reported on fiber burden in 10 cases of brake repair workers with mesothelioma. It is important to note that in this study elevated lung levels of

commercial (emphasis added) amphiboles in some brake workers suggest that unrecognized exposure to these fibers plays a critical role in the development of MPM.

Retrospective cohort studies of workers considered exposed to only chrysotile asbestos also provide evidence as to the inability of this fiber type to produce mesothelioma. Nine studies, purported to represent "chrysotile-only" exposed worker cohorts, are outlined chronologically showing the number of mesotheliomas and noting amphibole contamination:

Weiss 1977 264 workers No mesotheliomas

Robinson and Lemen 1979 3276 workers 17 mesotheliomas
Note: Exposure to both amosite (5 percent during three WWII years) and crocidolite (< 1 percent during the three WWII years). Other years amosite constituted 1 percent.

Rubino et al 1979 952 workers 1 mesothelioma
Note: The mesothelioma was not supported by histological examination and the exposures to what was termed "pure" chrysotile were reported as very high when both calculated and measured.

McDonald et al 1980 11,379 workers 11 mesotheliomas
Note: The study population worked in the Canadian asbestos mines in the towns of Asbestos and Thetford. As noted previously in this report, chrysotile asbestos from these mines had significant levels of the amphibole tremolite and tissue fiber burden/type analysis in the mesotheliomas from this population note the significant presence of tremolite in those individuals.

Acheson et al. 1982 570 workers 1 mesothelioma
Note: Exposure to crocidolite asbestos is possible as there was some interchange between another similar workforce where that fiber was used in gas mask manufacturing.

Dement et al. 1982 768 workers 1 mesothelioma
Note: Authors noted that only an "insignificant" quantity of asbestos fiber other than chrysotile was ever processed. Specific amounts were not mentioned.

McDonald et al. 1982 4137 workers 14 mesotheliomas
Note: Some amosite and a small amount of crocidolite were used in the Pennsylvania plant where the exposures occurred.

Newhouse et al. 1982 11,182 workers 10 mesotheliomas
Note: Eight of the ten individuals with mesothelioma had definite exposure to crocidolite exposure. One of the two remaining cases may have had "fringe" exposure to crocidolite and the other had previously worked at an asbestos cement factory. Crocidolite had been used in pipe manufacture and that worker's compensation case had been attributed to his earlier work.

Thomas et al. 1982 1970 workers 2 mesotheliomas
Note: Crocidolite asbestos had been used at the asbestos cement factory prior to 1936 and both individuals with mesothelioma had been employed there during the use of crocidolite.

In contrast to the hypothesis put forth by Smith and Wright in 1996, where the authors speculate that most mesotheliomas are the result of chrysotile exposure, numerous well-performed cohort, case-control and fiber burden/type epidemiological studies especially those involving considerable numbers of cases consistently show the presence of commercial and noncommercial amphiboles in mesothelioma cases, absence of mesotheliomas when discounting amphibole exposures and lack of increased risk of mesotheliomas especially in populations exposed to low levels of virtually pure chrysotile. The mere presence of chrysotile fibers, especially short ones, in an extremely limited number of "chrysotile only" cases is insufficient to causally associate chrysotile exposure alone with the development of mesothelioma. This is illustrated by "background" populations with numerous asbestos fibers of all types per gram of lung tissue (Dodson et al, 1991) and risk assessment studies showing no increased risk at such levels (see discussion below). It is clear that amphibole asbestos exposure can indeed produce mesothelioma at relatively brief and low levels of exposure. The mining/milling studies cited previously, support this point as even in populations highly-exposed to chrysotile fibers, mesothelioma is produced in those with brief commercial amphibole exposure and work in areas with tremolite fiber contamination.

It is also important to note that of the roughly 57 mesotheliomas that were reported in the "chrysotile only" cohorts, there was evidence for amphibole exposure in virtually all instances. In two cohorts there were no mesotheliomas as the tumor in the Rubino et al 1979 study was not histopathologically confirmed. Another critical point is that while there was no pathological fiber burden/type analysis performed in the cases of mesothelioma in these cohort studies, even the limited amphibole usage and expected exposures are consistent with the various assessments and reports of environmental and paraoccupational amphibole exposures as capable of producing mesothelioma at levels considerably lower when compared to occupational exposures. Yano et al, in 2001 reported a cohort study of 515 male asbestos workers who apparently worked with amphibole-free chrysotile and noted that heavy exposure (4.5 and 7.6 f/cc) to pure chrysotile can possibly cause lung cancer and malignant mesothelioma. A meta-analysis of 26 mesothelioma deaths from various studies reported an increase of mesothelioma in chrysotile-only exposed workers; however, there is no mention of potential amphibole exposure as discussed above (Lu et al, 2004). However, Burdorf et al (2005) noted the difference in pleural mesothelioma rates between Sweden and The Netherlands and proposed that the difference was likely explainable due to proportion of exposed individuals, exposure level differences and type of asbestos fiber.

Finally, risk assessment studies consistently show the lack of increased significant risk of mesothelioma at exposure levels likely to be the result of exposure to chrysotile asbestos from work with, and in and around asbestos-containing floor covering (Hodgson et al, 2000; Berman and Crump, EPA, 2003). There has been a wide range of risk assessments comparing the relative potency between chrysotile's and amphibole's ability to produce mesothelioma. Early

research discussed estimated differences of several-fold. However more recent risk assessments note a much larger difference. Hodgson and Darnton reported the following:

Crocidolite – 500 times more potent than chrysotile;
Amosite – 100 times more potent than chrysotile.

Berman and Crump in the 2003 draft EPA risk assessment conclude with a potency coefficient for chrysotile fibers that is 0.0013 times that for amphibole fibers.

There is significant scientific literature pointing towards a familial risk for mesothelioma (Ascoli et al, 1998; Huncharek et al, 1996; Ascoli et al, 2001; Hammar et al, 1989; Picklesimer et al, 2005; Bianchi et al, 2004; Risberg et al, 1980). However, certain genetic predisposition to mesothelioma development may make individuals significantly exposed to certain types of asbestos more likely to develop mesothelioma (Ohar et al, 2006; Bianchi et al, 1993; Heineman et al, 1996; Neri et al, 2005; Dawson et al, 1992).

Alternative Factors

As noted above, there are other more likely etiologies for Mr. Helfand's mesothelioma, primarily therapeutic radiation and amphibole asbestos exposure. Mr. Helfand has an employment history of "running wire" in homes during remodeling. The scientific literature does demonstrate that electricians were exposed to asbestos and were at increased risk of developing asbestos related disease (Paik et al, 1983; Peto et al, 1995; Bovenzi et al, 1993; Subramanian and Madhavan, 2005; Morabia et al 1992; Hodgson et al, 1988; Guberan et al, 1989; Menck and Henderson, 1976; Dambar and Larsson, 1987). It is noted that performing electrician tasks would potentially expose a worker to amphibole asbestos due to work in and around insulation in previously constructed structures. Boiler jackets and pipe insulation are specifically noted to be comprised of crocidolite and/or amosite asbestos in addition to chrysotile. Additionally, while working as a printer, Mr. Helfand described intermittently removing asbestos "lining" from a lead pot in a manner which would likely generate dust.

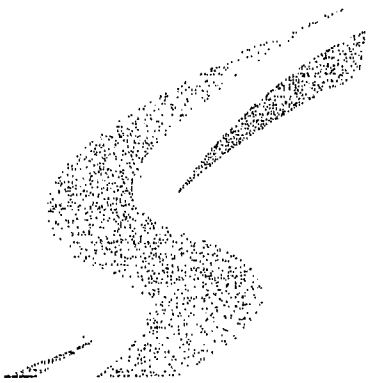
As noted above Mr. Helfand was treated with therapeutic radiation to his neck and upper back in 1986 for a reported six months. Radiation has been identified as a potential risk factor for various solid tumor cancers including mesothelioma in studies of radiation-exposed workers (Matanowski et al, 2007) and in survivors of the atomic bombings of Hiroshima and Nagasaki (see references). More definitive studies have been performed on large numbers of cancer patients who have received radiation therapy and subsequently developed second tumors (see references). Travis et al, in 2005 reported a 3.4 fold statistically significant risk for mesothelioma in a long-term group of 40,576 testicular cancer survivors who had received radiotherapy. Interestingly the mesothelioma risk for a latency of 10 -19 years following radiotherapy was six-fold and statistically significant. The methodology employed for this cohort study, its size and other strengths provide significant support for a causal association. Although not a statistically significant result, Neugut et al in 1997 reported a 56 percent elevated risk for pleural mesothelioma in a controlled study of over 250,000 women who survived breast

cancer and had been treated with chest radiotherapy. Mr. Helfand's approximate 19 year latency between his radiotherapy and his development of mesothelioma is consistent with these studies

Any opportunity to perform a fiber burden and type analysis on tissue in the case of Mr. Helfand may further help identify the most likely etiology of his mesothelioma.

SUMMARY

In summary, after careful review and evaluation of the case medical and other records, assessment of the current medical/scientific state of the art and my experience and training in the field of occupational and environmental medicine, it is my opinion that it is medically and scientifically more likely than not that while Mr. Helfand developed malignant pleural mesothelioma, there is no reliable scientifically-derived evidence that his work employing asbestos-containing floor tile covering products and any possible exposure to asbestos from such activities during the course of his activities at his own residences or during home remodeling, had any impact on the development or progression of his unfortunate disease. If indeed, asbestos-related, his mesothelioma is more likely than not the consequence of exposure to amphibole fiber exposures which reportedly occurred during work around pipe insulation, boiler insulation and other amphibole asbestos from other insulation sources. It is also more likely that Mr. Helfand's mesothelioma is of therapeutic radiation origin rather than being related to any exposure to chrysotile-asbestos from Kentile floor tile.



The opportunity to provide this assessment is appreciated. If there are any questions regarding this report or if additional assistance is desired, please do not hesitate to call.

Sincerely,

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